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PARALYTIC ILEUS

A Survey of the Etiology, Symptoms,
Diagnosis, and Treatment

Senior Thesis

1933

Victor Simecek

PARALYTIC ILEUS

The term "Paralytic Ileus" from a general standpoint is self-explanatory, however the exact mechanism is very much in dispute. In this paper there will be no attempt to point out any one definite opinion, but rather to survey the obtainable literature on the subject and present the more accepted views, allowing the reader to draw his conclusions as to the accuracy of each, both clinically and experimentally.

Before entering the subject proper, we shall briefly review a few phases of the normal intestine: Movements of the small intestine:--The movements of the small intestine may be classed as three types: (1) The Rhythmic Segmentation movements are brought about by localized rhythmic contractions of the circular muscle, generally in the duodenum and jejunum. These knead the intestinal contents, mix them with digestive juices, and spread them again over the absorbing surface of the mucous membrane. This process does not advance the material toward the lower bowel. (2) The Pendulum Movements consist of swaying movements, during which the contents of a short loop are thrown from one end to the other. Sometimes it looks as if the bowel was being drawn over its contents like a stocking over a foot. (3) Peristaltic Rush produces most of the forward movement of the intestinal contents. These are large waves which, from time to time,

run down the bowel. They may arise in any part of the small bowel and may run short or long distances.

Nerve supply:--The walls of the intestine are supplied by the sympathetic and parasympathetic systems, terminating in the nerve plexuses of Auerbach and Meissner. In addition, the sympathetic nerve with the plexuses act as inhibitory motor nerves of the stomach and intestines, while the vagus nerve supplies the exciting fibers to the digestive apparatus. It also contains some sensory conductors from the spinal fibers. With the sympathetic we link the splanchnics and with the parasympathetics the vagus nerve. In other words, we have an antagonistic dual innervation of the intestines by the vagus and splanchnic nerve fibers. Stimulation of the vagus nerves produces a motor effect, whereas stimulation of the splanchnics tends to produce an inhibitory effect. From this we can conceive how paralytic ileus could be produced by trauma through a reflex inhibition of bowel movements. It is from this standpoint that splanchnic and high spinal anesthesia offers such encouraging results in the treatment of ileus. The splanchnic analgesia interrupts the fibers of the splanchnic nerves at the point at which they break up into synapses which occur in the celiac or semilunar ganglia, i.e., the connection between the pre- and postganglionic fibers of the splanchnic nerves. In

other words, it tends to prevent impulses from reaching the intestines by way of the splanchnic, which would in turn give the vagus motor impulses full play. The celiac or semilunar ganglia, just mentioned, lie in the epigastric region of the abdomen behind the bursa omentalis and the pancreas, in the region of the origin of the celiac and superior mesenteric arteries. It is joined by the great and the lesser splanchnic nerves of both sides, by celiac branches of the right vagus, and by filaments from the upper lumbar ganglia of the sympathetic trunks.

Blood supply:--The intestines are supplied with blood primarily through the superior and inferior mesenteric arteries, and returns by the corresponding veins. This is of value only in that the increased tension of the gut may lead to interference of the blood supply to a portion of the gut, followed by gangrene and rupture.

To define the meaning of paralytic ileus offers considerable difficulties by reason of the fact that with ileus we understand a purely clinical entity including a great number of heterogeneous conditions, evident by disturbance in the intestinal passage. King (26) defines paralytic ileus as:--Perverted function of the intestine, prominently characterized by loss of tone of the intestinal musculature, of variable extent, and by extreme gaseous extension of the paralytic gut, fol-

lowing actual obstruction, circulatory failure, or toxemia; wherein there is defect of drainage per anum but maintenance of the excretory function of the liver and of the intestinal mucosa; wherein the content of the intestinal lumen becomes extremely toxic by reason of defective drainage and consequent putrefaction; wherein the oral rather than the aboral end of the alimentary tract is utilized inadequately as the excretory vent, wherein absorption of virulently toxic material from the intestinal lumen, and the prevalence of a paralytic intraintestinal gas pressure tend to perpetuate the stasis, and if the condition be not ameliorated, to kill. This with consideration of a reflex phenomena serve as a working basis.

ETIOLOGY:--

Adynamic or paralytic ileus may be divided into the following types:

1. Nervous origin---Spinal cord.

---Trauma to abdomen.

2. Infectious and Toxic origin---Peritonitis.

---Pneumonia.

3. Circulatory origin---Thrombosis.

---Embolism.

Injury to the spinal cord;---Injury to the spinal cord which results in paraplegia often produces an in-

intestinal paralysis which may be classed as a partial paralytic ileus. As a rule this is most manifest immediately following the injury and is sufficiently grave to be a contributing factor toward a fatal outcome. Gradually the intestine recovers its autonomic power of contractions and the ileus no longer exists. Transverse myelitis from new growth or degenerative disease may also cause serious stasis but the slowly developing lesion in this case usually permits the intestine to adjust its motor activity without aid of the central nervous system. The same statement applies to the disseminated and combined sclerosis of the spinal cord. In the absence of any central nervous injury or disease the intestinal musculature may exhibit a paralysis which is apparently due to disturbance of the splanchnic nerves. This is seen in renal and hepatic colic and in operative and other trauma not directly concerned with the abdomen. The mechanism of this form of ileus is not clear but apparently reflex in nature.

Abdominal trauma:--Often external trauma to the abdomen produces an intestinal stasis although there is not demonstratable injury within the abdomen. Usually this is temporary and passes away within a few hours, however it may be so severe that it is difficult

to rule out an intraabdominal condition. The latter is more frequently found in children.

Finally a form of ileus that probably belongs to the nervous paralytic type is not infrequently seen in cardiovascular disease and in debilitated persons of old age. In these conditions there is a gradual failing powers of peristalsis passing thru grades of constipation, obstipation and obstruction to a complete stoppage. There is considerable discussion as to the whether failing circulation or impaired innervation are the most responsible factors. The final stage, too, is usually hastened by an obstructive ileus caused by faecal impaction which the enfeebled gut is unable to propel forward.

Bacterial poisons:---Bacterial poisons are concerned in the production of the most serious and fatal forms of paralytic ileus. Such intestinal obstruction is one of the outstanding features of acute septic peritonitis. In fact it probably plays a most important role in the high mortality of peritonitis and many authors contribute death in that condition to the stasis of the intestine alone. One must, however, not lose sight of the role played by mechanical as contrasted with paralytic ileus in this connection. A peritoneum attacked by pyogenic bacteria rapidly form marked adhesions within its

component parts and such adhesions may readily result in obstructing the bowel by kinking, constriction etc.

Such a condition may also arise from the actions of bacterial toxins even though a peritonitis does not exist. The great tympanites seen in typhoid fever, pneumonia and pleurisy are expressions of this type of paralysis. It is questionable that these can progress to a complete ileus but the stasis may be severe enough to be an important factor in the clinical picture.

Embolism and Thrombosis:---The mesentric blood vessels are subject to invasion by emboli and are the seat of thrombosis exactly as in the case with other vessels in the body. Fortunately this is a rare accident because it is a particularly serious one. The vascular supply of the intestinal tract is largely a terminal one and hence the occlusion of any important vessel results in an immediate gangrene. There may be some question, therefore, as to whether the ileus that results should be classed under the paralytic group. The type of gangrene is exactly parallel with that seen in other parts depending on whether an artery or vein is the seat of the closure. In the former case the gut becomes paralyzed and then is subject to dry gangrene, whereas in the later the

condition is wet gangrene with bacteria playing an important part in the destructive process.

Contributions causes to the vascular paralytic ileus are those that produce similar changes in other parts; cardiovascular disease and septic foci with thrombo-phlebitis. There is no evidence that such foci in any one part of the body are more liable than others to find lodgement in the mesenteric vessels. It is worthy of note that focal infection within the abdomen, e.g., appendix, fallopian tubes, gall bladder, etc., are not especially prone to extend into important mesenteric vessels or liberate emboli which find lodgement there.

Murphy and Brooks (41), Hartwell and Hoguet (25), and others have presented evidence to show that the essential factor in the production of ileus is not the blockage of the lumen of the intestine but interference with the blood supply of the intestinal wall; only when such vascular disturbance occurs do the various phenomena characteristics of ileus develop. In their experiments with dogs they have observed the following:---Dogs with isolated obstructed duodeno-jejunal loop die without infection of the general peritoneal cavity or any evidence that the cause of death is any other than factors immediately associated with

the isolated obstructed loop. The symptoms which follow the operation are fairly constant. For the first twenty four hours after operation, the condition of the animal is not different from that after any laparotomy. Usually at forty eight hours, the animal appears drowsy and does not eat and vomites. The animal may rapidly become more apathetic and die in from seventy two to ninety six hours. Excessive vomiting or extreme loss in body weight are not necessarily factors in the symptoms. That symptoms are not the result of the loss of function of the duodenal and upper jejunum as the clear up after total excision of this part of the intestine. The content of the obstructed loop is a toxic fluid which, when introduced in comparatively small doses into the circulation of normal dog causes vomiting, fluid stool and death. Associated with the symptoms there is the characteristic pathological changes which consists in an acute hemorrhagic enteritis, especially marked in the duodenum and jejunum. The toxicity of the obstructed loop content is not destroyed by heating to 60 degrees C. until sterile, or even by boiling. The toxicity of the fluid is decreased several times by filtering through a Berkefeld filter. The amount of filterable toxin is increased by pro-

longed autolysis, and in no case did the animal show symptoms without demonstrable change in the mucosa.

In another series of experiments these changes could be demonstrated by isolated ileus obstruction.

They then isolated, obstructed loops with interference to the blood supply. They found animals with obstructed segment of bowel in which there is a disturbance in the blood supply develops symptoms more readily and die in much shorter time than animals with obstructed segments of bowel in which there is no nutritional disturbance. Also the fluid accumulating in segments of bowel with vascular disturbance, especially arterial obstruction, is very toxic in a period of less than twenty four hours, at which time an obstructed segment of bowel without vascular disturbance contain practically no content. The Berdefeld filtrate from the contents of an obstructed segment of bowel with venous obstruction of twenty four hours or less duration, may be injected intravenously in healthy dogs in comparatively large dose, without causing symptoms.

In an another series of experiments to contrast the amount of secretion from the jejunum with that from the ileum, and to show the influence of the

difference in the amount of secretion in the production of symptoms, they found that isolated, drained loops of jejunum discharge constantly a relatively large quantity of a thin fluid, while isolated, drained loops of ileum discharge very little. Also following the closure of such jejunal loops, the animal develops in the course of from seventy two to ninety six hours, symptoms of grave intoxication associated with marked distention of the occluded loop and an accumulation of a toxic content. The enterotomy of an isolate, drained loop of ileum may close and the loop may be completely occluded for long periods of time without signs of intoxication. Such a closure is associated with very little and a gradual distention of the bowel. The contents of such a loop may be very toxic with the animal showing ~~no~~ signs of intoxication. Any operative procedure leading to damage of the mucosa results in symptoms.

To prove that the symptoms are due to a toxemia and to illustrate the effect of strangulation or distention of the intestine on the absorption of the toxin the following results were obtained:---Following occlusion of the veins to a segment of bowel, there is immediately hemorrhage in the lymphatics. There is also very quickly and ex-

tensive distruption of the mucosa and hemorrhage into the lumen of the bowel. Similar changes follow rapid distention of the bowel from increased pressure within the lumen. Also with the introduction of the toxic contents of an obstructed, intestinal loop into the lumen of the bowel and the occlusion of the veins of loop containing the toxic fluid, the fluid from the thoracic duct contains a toxic substance which when given to a normal dog, results in symptoms similar to those following the injection of the toxic loop content.

With gallbladder obstruction it was found the steril gallbladder of the dog can be obstructed and the blood supply interfered within the point of gangrene without the animal showing noticeable symptoms. Also following obstruction and interference of nutrition of the gallbladder containing the bacteria of the intestine, the animal shows symptoms similar to those of intestinal obstruction.

In experiments on a dog in which a drained jejunal loop became steril they found the secretion of a well draining jejunal loop, after complete healing of the operative trauma had no toxic manifestations. After continued drainage a jejunal loop may become sterile, in which case complete occlusion may persist

for a period longer than is consistent with life ~~with~~ similar occlusion of such loops containing bacteria, without the animal showing symptoms and without the accumulation of toxic content. Also after infection of the isolated loop, continued obstruction leads to symptoms and the accumulation of a toxic content.

Toxic substance may also be produced by the incubation of an intestinal segment which produces symptoms, death and the same pathology as are produced by the toxin from an obstructed intestinal loop, as shown by injections into dogs.

From these experiments the following conclusion was drawn:---

1. In intestinal obstruction, the content of the obstructed bowel contains a toxin which, when absorbed in sufficient amount, produces definite symptoms and pathologic lesion and death.
2. The toxins are the result of bacterial growth. They are not specific for any part of the intestinal tract, and may be found in the gallbladder.
3. The chemical and physical characteristics of the toxic substance may vary with the length of time which the obstruction has existed as well as with the different conditions under which the ob-

struction occurs.

4. This toxin may enter the circulation by way of the thoracic duct.

5. Death after intestinal obstruction is the result of a toxemia which may be independent of infection of the peritoneal cavity of general circulation.

6. This toxic substance does not pass through a normal mucous membrane.

7. In the production of symptoms the factors which make this absorption possible are more important than the factors which produce the toxin.

8. Interference with the circulation of the obstructed intestine is an essential factor in allowing this abnormal absorption.

9. Simple obstruction of a segment of duodenum or jejunum results in earlier and severe symptoms than similar obstruction of a segment of ileum because the secretion into the lumen of the former leads to rapid distention and circulatory disturbance in the bowel wall.

10. The symptoms and pathologic lesion following the intravenous administration of the contents of a segment of bowel after obstruction are the same as those described resulting from intravenous

injection of certain of the ptomain poisons.

11. In the surgical treatment of cases of intestinal obstruction, that part of the intestine with a mucous membrane which has been so damaged as to permit of abnormal absorption should be resected rather than drained.

An attempt has been made recently by Owing, McIntosh, Stone and Weinber (50) to explain interference with the blood supply on the basis of increased intra-intestinal pressure. Though these particular investigators found the pressure in various parts of the intestinal tract to be similar regardless of the site of obstruction and therefore were unable to explain certain characteristic differences between "high" and "low" obstruction.

Dragstedt, Land and Millet (15) have attempted to incriminate the anatomical topography of the intramural blood supply as of direct etiological significance in the development of ileus. They believe that increases of intra-intestinal pressure are responsible for circulatory embarrassment, but that the degree of embarrassment caused by a given increase in intra-intestinal pressure is dependent upon relative length of the veins coursing through the intestinal wall; the intramural portion of the veins is longer in the

upper intestinal tract and is occluded much more readily than the intramural portion of veins which occur more distally and are shorter.

In postoperative cases of paralytic ileus, such as frequently found following a clean case of appendicitis, or rarely following a difficult delivery, where infection is not present it is undoubtedly on a nerve disturbance basis. A case has been reported following a severe blow on the chest fracturing three ribs. Many cases are probably due to rough handling, actual injury to the bowel, extensive manipulation, and the use of too hot or cold sponges. However Cannon, and Murphy (9) in a series of experiments have shown the lack of intestinal movement is most marked when the intestines are roughly handled. Hot and cold air exposures did not cause a very noted disturbance.

King (26) in a series of experiments on intestinal inhibitor reflexes has concluded that:---

1. Impulses from the urinary tract, rectum, peritoneum and certain skin areas reflexly diminish tonus and movements of intestines.
2. Splanchnics contain the efferent paths for these reflexes.
3. Vagi very little, if any, involved in these reactions.

4. Some efferent paths involved are the hypogastrics.

5. Relation of disturbance of gastro-intestinal tract^{to} conditions in pelvis.

Mueller(40) gives considerable stress to infection as a definite etiological factor. In the course of a local infection in the abdominal organs there develops, as the result of an autonomous-vegetative reaction, a dilatation of the vessels in the splanchnic area with a corresponding reactive withdrawal of blood from the periphery. As a result of this disturbance in the distribution of the blood, there is a change also in function in the splanchnic region and the periphery. Synchronous with the distention of the vessels, there is reflex stimulation of secreting cells in the stomach and intestines. This is followed by the increased production of concentrated gastric juice, mucous and bile with distention of the gastro-intestinal tract which explains a series of typical ileus manifestations such as discomfort, nausea and vomiting.

Also the paralysis of the bowel in the early stages of peritonitis is not to be regarded as a muscular paralysis. It is the result of the increased vegetative stimulation which leads to an increase in the

voluma and elongation of the smooth musculature. In contrast to striated muscle, which reacts to stimulation with the characteristic rhythmic contraction, this capacity for contraction is absent in smooth muscle. Peristaltic movements which arise simply from shrinkage of the intestinal musculature cannot occur as long as the condition of increased voluma persists. Consequently the clinical picture of intestinal paralysis results from the excitation state in the aplanchnic area caused by the peritoneal infection. Under physiological conditions, the transmission of vegetative stimulation leads to segmental relaxation of the affected portion of bowel. Contraction occurs only when the irritating impulse ceases, as the reaction of the previously stimulated intestinal musculature. If the irritation continues, as is the case in peritoneal infection, intestinal contractions do not occur and the bowel remains in a relaxed and dilated state.

The fundamental cause of death in intestinal obstruction is still under dispute. However it is generally accepted that the toxemia holds the most prominent place. This causes a destruction of protein and diminishes kidney activity, so that nitrogenous bodies accumulate in the blood stream. The kidney in-

volvement may be impaired further by dehydration and cardiovascular changes ending in shock. Contributing causes of death are the alkalosis, produced by hypochloremia, leading to a derangement in the carbohydrate metabolism. This factor seems further effected by limited food intake.

Haden and Orr (23) - (24) in a series of detailed experiments of dogs, rabbits and monkeys, have called attention to certain quite constant and characteristic chemical changes taking place in the blood of animals particularly dogs, after experimental high intestinal obstruction. Soon after the obstruction is made in the upper jejunum, the blood chlorides begin to fall, high level of non-protein nitrogen, suppression of chloride, excretion, the carbon dioxide combining power of the blood, increases; and when the blood chlorides have been depleted about one-fourth, there is a beginning rise in the blood urea nitrogen. ~~While~~ the vomiting that occurs there is of course some loss of chlorides, but they feel that this cannot be the sole cause of chloride depletion, since the same results occurs in rabbits which do not vomite. These facts lend great credence that the chlorides are being used up or fixed in the tissues, and are used by the

organism in a protective role. To prove this they have shown it is possible to keep dogs with high intestinal obstruction alive for twenty-eight days without the development of toxemia or typical blood chemistry changes, and then to see them die of pure exhaustion and starvation. Control animals die in from two to six days; dogs given distilled water in place of the chlorides die even sooner. No other salts tried have shown the same protective mechanism. Later on similar obstruction experiments were repeated, doing a lateral anastomosis on about the third day or after the toxemia had developed from the primary obstruction, and all eight dogs died in from one to nine days. Six dogs were given distilled water and all died. One dog was given daily one-half per-cent sodium chloride subcutaneously, lived thirty days, and died of exhaustion without any toxemia developing.

Source of gases of the intestinal tract:---

Most writers agree that the gases commonly present in intestinal meteorism are carbon dioxide, oxygen, hydrogen, methane and sometimes hydrogen sulphide, the proportions being subject to considerable variation. The origin of these gases may be listed as:--

1. Decomposition of intestinal contents.

2. The diffusion of blood gases into the intestinal lumen. The walls of the intestine are permeable to gases, and an active interchange takes place, tending to keep the gases on the two sides of the intestinal mucosa in equilibrium.

3. The gas normally found in the stomach consists primarily of atmospheric air, admitted by swallowing. Under some conditions the amount of air is considerably increased, and hence it is probable that its passage downward into the intestine may be an important factor in the production of distention in certain cases.

The body eliminates intestinal gases by:--

1. The expulsion of gas by motor activity is an important method of eliminating abnormal accumulations which sometimes follows injury ^{which} is a cardinal factor in distention.

2. There is also the reverse process of the blood gases into the intestine, the absorption of intestinal gases into the blood stream. Experiments have shown the amounts that can be eliminated are considerable. Interference with the circulation hence plays a very important part in the presence of gas in the intestine.

Mo Iver, Benedict, and Cline (34) lay con-

siderable stress on the part that swallowed air plays in many cases of postoperative ileus. A study of 107 cases in which a laporotomy had been performed, distention was present in 36. The highest incidence was found in operation on the biliary tract. The anesthetic employed except in a few cases, was ether, preceded by nitrous oxide. The length of operation did not apparently influence the incidence of distention. With another series of 25 cases they used a stomach tube. While the patient was still under ether, a small stomach tube was passed into the stomach through the nares. The gaseous contents of the stomach were then drawn off by means of a syring; the tube was left in position for from twenty-four to forty-eight hours, and the gastric contents aspirated twice daily. In this series only three cases ~~were~~ distention.

SYMPTOMS AND DIAGNOSIS:

The early symptoms and clinical features which are regarded as important are as follows;

1. Distention:--The abdomen should be observed in a good light and completely exposed. The distention is always present and becomes progressively worse as the condition continues.
2. Tympanism:--Accompanies distention.
3. Early dehydration:--The mouth is dry. The tongue is parched and brown. The patient complains

of thirst, though he may be unable to retain any liquids taken per mouth. There is a frequent desire for orange juice or ice water.

4. **Facies:**--The patient's expression is a peculiar one in that the eyes are bright. The cheeks are flushed and there is a look of anxiety stamped upon the individual's face. This depends also upon the associating condition. If accompanied by severe peritonitis or a mechanical obstruction the facies are more marked.

5. **Peristalsis:**--Not present in the paralytic type, and though the abdomen be sharply distended one does not hear the gurglings and tinklings due to the movements of the intestinal contents. However in the bacterial types such as peritonitis, the intestinal movements may be heard in the earlier stages.

6. **Clapotage:**--May be present, but not in all cases.

7. **Pain:**--When not associated with infection, the patients not, in many instances, complain of severe or recurrent attacks of pain. However in the bacterial type there are acute, recurrent attacks of severe, colicky pains. This may be local or general according to the associated condition.

8. **Nausea:**--Present and constant.

9. **Vomiting:**--At first is foul, putrid, active,

bilious vomiting; it may be black from altered biliary excretions following ether anesthesia. In the graver cases it tends to become more and more offensive. Later if the patient lives long enough it becomes black and copious. Not a projectile type but more of a spilling over of the mouth.

10. Hiccough:--Is often present and is of interest as it is associated with heart sign. With the stethoscope on the heart apex, the hiccough is synchronous with the beginning of extraordinarily powerful vagal arrest of the heart in diastole, which may persist several heart cycles.

11. Twitchings of the fingers and face, even to the typical contraction of tetany. They have a lethargy and twitching somnolence.

12. Temperature:--Is scarcely increased until toxic products have begun to be absorbed and made themselves manifest, or unless accompanied by infection.

13. Pulse:--Is accelerated and in the absence of corresponding temperature rise is highly significant. The thready pulse of the patient in shock may be present.

14. Intolerance of the stomach for cathartics.

15. Futility of enema.

16. Laboratory findings:--Lowered blood chloride,

high carbon dioxide combining power, plus urea nitrogen retention.

Further discussion of symptoms will be taken up in the diagnosis and case reports.

In the diagnosis of this condition, the discussion will be limited principally to a differential diagnosis between the mechanical and paralytic types of ileus.

Auscultation of the abdomen is, in the majority of cases, the most accurate diagnostic feature. One can elicit exaggerated peristaltic sounds and the recoil of trapped gas and fluid in the mechanical obstruction with ease, while in the paralytic type the abdomen is quiet.

Barium enemas or barium by mouth should not be relied upon to make the diagnosis. Barium by mouth is dangerous---by enema---it reveals nothing except that the obstruction is in the large intestine and the technique of administration causes delay and loss of valuable time. One may however learn much by an X-ray plate.

Mendonca, (37), lists the following differential diagnosis:--

DYNAMIC ILEUS

MECHANICAL ILEUS

ANAMNESIS

Previous existence of a nervous condition that may cause spasm or intestinal paralysis. Severe pain sufficient to cause a deep alteration of the nervous centers. Infectious diseases, such as peritonitis, meningitis, pancreatitis, appendicitis, etc. Intoxication and poisoning (spastic) Absence of any previous and local lesions that might cause of mechanical obstruction.

FACIES

Depending on the disease that caused the obstruction; abdominal in generalized peritonitis, twisting of pedicles, etc., and generally normal in local peritonitis without severe pain, salpyngitis and appendicitis without great repercussion of the peritoneum etc.

Constipation or diarrhoea, associated with tympanism and local pain. Existence of a lesion that, by its evolution, may cause obstruction.

Previous existence of hernia or calculous cholecystitis.

Swallowing of foreign bodied that may obstruct the intestine.

As a rule normal during the first forty-eight hours and altered after this except in high obstruction of the small gut, strangulation, and peritonitis as an initial complication when the facies is soon altered. In rare cases of low obstruction we may observe a composed facies for seven or more days.

PULSE

The same as in the disease that caused the ileus. A low tension, without general peritonitis, intoxication, or violent pain, is an exceptional condition.

If there is no strangulation volvulus, or other lesion causing a deep alteration of the nervous system, the pulse is normal or almost normal and of good tension, but as the infection progresses the pulse become rapid and of low tension.

GENERAL TEMPERATURE

The same as the disease that caused the ileus.

Normal at first. It rises progressively as the intoxication increases. When peritonitis is a complication, the temperature is high or low as is the case.

LOCAL TEMPERATURE

Is dependent on the disease that caused the ileus.

Normal as a rule.

VOMITING

Uncommon but frequent and resistant to treatment when there is peritonitis. Never faecaloid, if there is no peritonitis.

The frequency and type depend on the height of the obstruction.

TYMPANISM

Permanent or not, it is found in all cases of obstruction. The presence of gas in the peritoneal cavity is rather a sign of perforation of the stomach or intestine.

Always present. Increases progressively as time passes, and decreases only by belching.

ABDOMINAL PAIN

Absent, spontaneous or provoked, local or general, according to disease causing ileus.

Pain only during contraction of the intestine to overcome the obstacle or when peritonitis complicates the obstruction.

RESPIRATION

Varying according to the diseases that caused the ileus.

If there is no severe pain, at first normal; in peritonitis or after great distention of the abdomen breathing becomes thoracic, superficial and frequent.

PERISTALSIS

Increased in spasmodic ileus and diminished or abolished in the paralytic type.

Always increased and associated with borborygms audible at a distance; de-

creased or absent when the ileus is complicated with peritonitis or the disease is in the last period

SHAPE OF ABDOMEN

More or less distended according to circumstances.

The abdominal wall is uniformly distended; but the presence of greatly distended loops or tumors may cause protrusions.

DIURESIS

If the diseases that caused the ileus does not present special characteristics; diuresis is in the inverse ratio to the increasing intoxication and frequency of attack of vomiting.

Grows less and less, if the obstruction is not removed; the urine becomes concentrated and nephritis appears.

PERMEABILITY OF THE INTESTINE

Restored when the disease that caused the ileus is cured.

Restored when obstruction is removed.

TREATMENT:

PROPHYLAXIS:--In the postoperative cases, prophylaxis should always be the watchword. Many cases of postoperative ileus have been brought about by too rough handling, or prolonged operations.

Cannon and Murphy (9) in a series of experiments on dogs have found that where the tissues are manipulated a great deal both by hand or with gauze, there is a cessation of all intestinal movements. They also found that exposure to air, both warm and cold, although diminishing the intestinal movements slightly, did not stop them completely. However it is evident that these conditions may be contributing factors and should be avoided as much as possible. Also the use of too hot or cold towels maybe contributory factors in the production of paralytic ileus. Badly given anesthesia, great loss of body fluids, and the leaving behind of raw surfaces within the abdomen are also factors. Guthrie (22) described a method of Trendelenberg anesthesia. The sole object of the method is to prevent the use of gauze packing in pelvic surgery. The anesthetic is begun with the patient in high Trendelenberg position. By the time the abdomen is opened, the pelvis will be practically free of all loose intestinal coils, so that it is often only necessary to use the end of a square piece of gauze in the upper angle of the wound to obtain adequate exposure.

The use of spinal anesthesia has the advantage in

carefully selected cases, of giving complete relaxation of the abdominal wall and the collapsed state of the small intestine which permits complete exposure with the use of the smallest amount of gauze and truma to the visera.

Due to the loss of body fluids and depletion of chlorides, saline solution should be given immediately. Where possible, it is also desirable to make a determination of the blood chlorids as well as the carbon dioxide combining power of the plasma, since the procedure is a guiding feature in the postoperative treatment of the case.

Sorese (56) suggests stretching of the anal sphincters as a preventive measure. This is based on the grounds that if the intestine is slightly parietic and gas forms into its lumen, this gas will stimulate the sympathetic impulses and establish a vicious circle. This condition is aggravated by the fact that the anal sphincters are contracted and prevent the escape of gas. Stretching puts them in a parietic condition allowing the escape of gas. The dilation should be made gently and should produce as complete a relaxation of the sphincters as obtained previous to a radical hemorrhoidectomy. There are practically no contraindications to this procedure.

Williams (60) believes there are clinical resemblance of the toxemia in acute obstruction and peritonitis with ileus and the toxemia of gas gangrene, and that is presence of the actual exotoxin of *B. Welchii* in the

content of small intestine of cases of acute obstruction and peritonitis. He shows a decreased mortality in clinical cases where the B. Welchii antitoxin was used, and recommends it be given, as does Southam (57) and others, before operation and daily after operation till bowels normal.

Over purgation before or after operation should be avoided so as not to cause excessive inflammation of the intestines.

IMMEDIATE:---The immediate treatment of paralytic ileus depends a great deal on the condition present and the individuals preference. Many types of treatment are suggested in literature and each have their merits.

1. Elimination of cause with as little manipulation or trauma as possible.

2. Maintenance of position (Fowlers)

3. Heat to abdomen. This reduced deep congestion and stasis in the intestines by peripheral dilatation.

4. Constant gastric and duodenal drainage and lavage. This should begin at the first sign of distention and continued until the tone of the bowel is restored. Should be washed frequently---every hour or two--- and a Refuss tube left in situ between the lavages, and getting the tube into the duodenum, thereby permitting it to drain away the accumulations with the idea of ridding the patient of toxic products that may accumulate through reverse peristalsis.

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(5) The efficacy of drugs, such as castor oil, calomal, pituitary extract. etc., are no doubt of some benefit used to supplement other treatment but their action depends on contraction of the bowel. If the bowel is paralyzed, these drugs will fail to act and will add to the strain on the heart, already damaged by toxemia. Ochsner, Gage and Cutting (44) in a series of experiments on intestines of dogs found that the following drugs are of very little value:--

Pituitin; There was a initial ~~rise~~ in blood pressure with a subsequent lowering below normal with a following rise to higher than previous. The tone and peristaltic waves of the intestines were decreased.

Physostigmine; The blood pressure here was increased as was the tone and amplitude of contractions, although the later was slight.

Choline; Showed an increase in blood pressure. The action on the intestines was inconstant and insignificant.

Pitocin; Showed no effect on the blood pressure. The intestines showed variable effects with no consistency.

Peristaltin; No effect on blood pressure. On the intestine was either nil or in the direction of a decrease.

Sodium Chloride; Showed a variable effect on

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blood pressure. The intestines showed slight increase in tone and amplitude of peristaltic waves.

With these results the authors conclude that the value of drugs has been considerably over-rated by clinical observation. Pituitin, then, not only does very little good but may do harm by its depressive effect, and although in the majority of cases it increased the blood pressure, it may also decrease it. Physostigmine, was the only drug in their opinion that acted with any consistancy and this action was usually slight. Cholin, pitocin and peristaltin all proved very disappointing as shown above.

Although the above is throughly experimental data, it would seem that the drug therapy rests on a rather precarious foundation. However in selected cases, such as the earlier cases, these drugs may be given with other treatment and will be of some benefit. Many clinical men urge the use of pituitin quite strongly.

6. Morphine:--For a long time most authors have looked upon morphine administration in intestinal atony with considerable doubt. Plant and Miller (52) were among the first to do any authorized work on this subject. They found that morphine increased muscular tone, frequency and amplitude of peristaltic waves, and amplitude of rhythmic contractions both in dog and man. This is also true of some other opium alkaloids only

larger doses were required. Later Dvorak (16) and others obtained the same results with morphine and demonstrated that intestinal borborygium which may be heard with the stethoscope early in intestinal obstruction are not silenced by the administration of the drug. Added to this morphine is indicated to give the patient rest between the interval of a treatment so^{as} to conserve his strength, which of course is a very vital factor.

7. As suggested previously the administration of B. Welchii antitoxin, first brought to the literature by Williams (60) may be attempted. He reports his mortality decreased in appendicitis operations followed by paralytic ileus from 6.3 percent to 1.17 percent and in intestinal obstruction from 24.8 percent to 9.3 percent. These results have been confirmed by other authors but considerable work need be done on the subject to prove its value.

8. Administration of chlorides:--The benefits of chlorides are three-fold;

- a. Replenishment of depleted chlorides.
- b. Retardation of absorption of toxic bowel content.
- c. Stimulation of active peristalsis.

Since the work of Haden and Orr and others, hypertonic saline injections, especially in paralytic ileus has taken a prominent place in the treatment of

this condition. It was found in numerous blood studies that the chlorides were markedly decreased. In experimental studies in dogs, the dogs given chlorides were found to live much longer than those without the chlorides. Clinically many investigators have found that in very alarming cases of paralytic ileus, the injection of hypertonic saline solution relieved the symptoms, with a stopping of vomiting and followed by the escape of gas. This action is explained as causing in a direct manner a strong contraction of the smooth musculature, and on the other hand it evokes a strong afflux of tissue fluids into the blood, and thereby makes possible the elimination of a number of toxic substances from the body which have accumulated in the tissues. This detoxicating action of the hypertonic saline solution is regarded by many as still more important than the stimulating effect upon the peristalsis. Administration of the solution varies with the severity of the condition and method preferred by the operator. Most authors proceed as follows:--

The operated patient is treated with heat and the simultaneous introduction of an intestinal tube, when no gases have escaped after twenty-four hours. In the mild cases the impaired intestinal function is thereby stimulated. A simple enema in the form of a glycerine clyster than produces the evacuation of the contents of the ampulla. When the intestine after thirty-six hours has not resumed its

function, in spite of these measures, 10-20 c.c. of saline solution should be injected intravenously. In the mild cases an elimination of gases is attained in a very short time after the injection. When the desired success remains absent, the patient receives a rectal infusion of a twenty percent saline infusion in a quantity of about 125-150 c.c. This is best given in the form of a drip-enema. In severe cases, a combination of both may be used from the onset, and the amounts increased up to the tolerance of the patient.

Numerous authors reported cases treated with hypertonic saline solution with very encouraging results. Kuesenhoff (28) reports a case of severe paralytic ileus resulting from a mild pyelitis:--

A woman 46 years old, who on admission presented a complete severe paralytic ileus. Of the history it is mentioned that the patient suffered from a chronic obstipation and had suffered from gastric colics allegedly six weeks prior to admission to the clinic. Five days prior to admission she became acutely ill during the night with severe chills. On the following day she suffered from a severe feeling of illness and pains around the waist. Her temperature rose to 102.6. Stools were only attained by means of purgatives. The urine examined by her family physician contained leukocytes and epithelial cells. On the second day following the initial

chills, severe abdominal pains and complete retention of gases and stools set in. The abdomen was distended. The patient vomited. Enemas were unsuccessful. The abdomen remained distended and exceedingly painful, especially the epigastrium. Micturition was possible, but connected with a burning sensation. The condition of the patient became progressively more alarming, so that the attending physician referred her to the clinic on account of ileus. The author found the woman in very poor condition with distinct facies abdominalis, high fever, and a dry, coated tongue. Pulse accelerated. The abdomen was very much distended and painful to pressure. Tenderness in left renal region. Intestinal sounds were not perceivable. The urine contained leukocytes. Immediately following admission the patient was given 15 cc. of a 10 percent saline solution intravenously as well as a rectal infusion. Already within the first hour considerable gas and fecal matter were eliminated. The alarming abdominal symptoms disappeared and the intestine again functioned normally. The urological examination made a few days later revealed a mild pyelitis of the left side, which was regarded as the cause of the severe ileus, since later examination of the gastro-intestinal tract showed no other reason therefore. The prompt results of the author's therapeutic measures showed that an accompanying ileus was involved, the pyelitis being the cause.

9. Splanchnic and Spinal Anesthesia:--This treatment is based on the antagonistic dual innervation of the intestines by vagus and splanchnic nerve fibers; stimulation of the vagus nerves produces a motor effect whereas stimulation of the splanchnic nerves produces an inhibitory effect. Any procedure, therefore, which tends to prevent impulses from reaching the intestines by way of the splanchnic, tends to render inoperative the inhibitory system and leave the motor supply in full control. Splanchnic anesthesia interrupts the fibers of the splanchnic nerves at the point at which they break up into the splanchnic plexuses anterior to the bodies of the first and second lumbar vertebrae. Nicotine splanchnic analgesia interrupts the synapses which occur in the semilunar ganglion, i.e., the connection between the pre- and postganglionic fibers of the splanchnic nerves. Spinal anesthesia should be as effective as splanchnic analgesia, if all the fibers entering in the formation of the splanchnic nerves or at least the reflex involved in the splanchnic control of the intestines are blocked. That spinal analgesia is actually not as efficient as novocain splanchnic analgesia in experimentally produced ileus seems to indicate that a part of the reflex involved in inhibitory regulation of the intestinal movements occurs by way of reflex arc which does not transverse the spinal cord.

Splanchnic analgesia is a method which should be used early and not as a last resort, since it could hardly be expected that a patient who has already absorbed a lethal dose of toxin can be revived by any means at all. Also accessory treatment, of course, should not be neglected.

There are three methods of giving splanchnic analgesia:--

a. Wendelung technique:--Involves the puncture of the anterior abdominal wall, and probably has no place in rational surgery at all

b. Braun technique:--Involves a preliminary laparotomy and somewhat extensive intra-abdominal manipulation, and is, therefore, practically never indicated in the treatment of ileus.

c. Kappis technique:--Involves the introduction of a needle posteriorly through the flank is almost invariably the method of choice. There are four points of injection; one on either side approximately opposite the first lumbar vertebrae and one on either side approximately opposite the second lumbar vertebrae. The agent usually used is the plain two percent novocain solution. Twenty cubic centimeters of the solution should be injected at each of the four points. Due to the inhibitory effects of adrenalin, ephedrin, and atropin on intestinal movement, these drugs should never be used before, with, or

after splanchnic analgesia in the treatment of ileus. Fall of blood pressure is invariably found after successful splanchnic block, and although undesirable is not a contraindication for this form of therapy. Since the effect of splanchnic analgesia on the motility of the intestine is only transitory, lasting from six to thirty minutes, this procedure may be repeated one or more times in case the previous injection has not proved altogether successful after a lapse of as short a time as one hour.

Spinal anesthesia is also used by various authors. Brunn and others (6) found it relieved the acute condition following operative procedure in 60 percent of the cases. He believes that best results are obtained by this method of treatment in postoperative paralytic ileus, and that where relief is obtained in such case a secondary operation is not necessary. In all other cases he believes that spinal anesthesia should be used only as an anesthetic and not a method of treatment. He reports the following case developing from a megacolon and relieved by spinal anesthesia.

L. B. age sixteen years, female, entered the Mount Zion Hospital on the surgical service of Dr. Harold Brunn on January 25, 1928. Her entry complaint was abdominal distention and constipation. There was no history of any operations. The mother of the child stated that since

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early childhood the patient had been markedly constipated and on numerous occasions had had attacks of abdominal distention. They were usually relieved by enemata and cathartics. A month previous to the present entry the patient had an especially severe attack of constipation with abdominal distention which was not relieved by the usual enemata, but after three days an exceptionally large dose of castor oil produced an evacuation with complete relief of the distention.

Three days previous to the present entry, patient again became markedly constipated and distended. Castor oil and enemata were tried at home repeatedly without success, and as the patient's distention was increasing she was brought to the hospital. The general physical examination showed an apathetic girl, exhibiting apparent signs of endocrine dystrophy. Her stature and mentality were much below that of a sixteen-year-old girl. Her facies were typical cretinoid, and she had the male distribution of hair, dry skin, abnormal distribution of fat pads and spad-like hands. The physical findings otherwise were negative, except for the extremely distended abdomen. The distention was so marked as to cause protusion of the umbilicus, and palpation was impos-

ible because of the tenseness of the abdominal wall. No peristalsis could be heard. Temperature on admission was 36.4 c. Pulse was 80. Hemoglobin, 75 per cent; red blood count, 4,900,00; white blood count, 12,700, with 87 per cent polymorphonuclears.

Course in Hospital.--Aside from the pain caused by the tremendous distention along both costal margins the patient had no complaints and did not look toxic. It was decided to attempt to relieve her distention by gas enemata and pituitrin. On January 25, the day entry, a 1-2-3 enema of glycerin, magnesium sulphate and water, preceded by one-half cubic centimeter of pituitrin, was given without results. On January 25 a milk of molasses enema was given with the patient in knee-chest position, the enema being preceded by one-half cubic centimeter of pituitrin. Again, no results were obtained. Later on this day an olive-oil enema was given to be retained. And on January 27 three doses of one-half cubic centimeter of pituitrin one-half hour apart were given, and a small amount of gas with slight amount of liquid fecal matter were passed, but without any relief of the distention. On this day, for the first time, the patient vomited on two occasions, and her temperature for the first time was slightly above normal, it being 37.4. Pulse,

however, remaining at 80. The vomitus was not fecal and consisted mainly of bile and gastric mucus, patient having had nothing by mouth since her admission to the hospital.

On the morning of January 28 patient's distention seemed more pronounced than at any time since her admission to the hospital. It was a week now since the distention was present, and in that time there had been practically no bowel movement or passage of gas. It was thought that something of an operative nature would have to be done. The patient was not showing any marked temperature reaction or increase in pulse rate, but we did not feel that much reliance could be placed on these usual indications of intraabdominal pathology because of the endocrine peculiarities of this patient, nor did we wish to wait until fecal vomiting had commenced before interfering. It was decided to take the patient to the operating room, induce spinal anesthesia, and if no relief was obtained by this method to perform a laparotomy.

Spinal anesthesia was induced by the use of .6 of a gram of neocain injected intraspinally between the twelfth thoracic and first lumbar vertebra. Five minutes after the injection of the neocain the skin anesthesia extended from the toes to the level

of the nipples, showing that we had a high and complete intraabdominal anesthesia. Ten minutes after induction of the anesthesia the patient spontaneously passed a considerable amount of gas, and in the succeeding twenty minutes, aided by gentle abdominal massage, tremendous amounts of gas and soft fecal material passed, the abdomen becoming flattened out in a manner almost comparable to the flattening of a toy balloon after it is punctured.

Subsequent Course in the Hospital.--The patient has no ill effect from the spinal anesthesia and with the aid of colonic flushes in the next couple of days a tremendous amount of fecal matter was passed by the patient. From the third day after the spinal anesthesia to the present time, patient has had voluntary bowel movements with the aid of simple cathartics and mineral oil, and there has been no recurrence to date of the obstruction.

Subsequent studies of this patient by x-ray showed her to have a marked enlargement, elongation and dilatation of the entire colon. The sigmoid, in particular, was elongated and enlarged, and the x-ray diagnosis was that of megacolon. The gastrointestinal fluoroscopy otherwise was negative. The basal metabolism also confirmed the impression of cretinism showing basal metabolic rate minus 35 percent.

10. Enterostomy:--Any operative procedure in cases of paralyzed bowel only drains the effected loop and is hence of limited value. It does, however, have its place principally where toxic symptoms are manifest quite strongly. Care should be taken however, and if the patients condition is too critical, an attempt should be made to strengthen his condition before operation. Also constant drainage may end fatally if carried too far resulting from the loss of intestinal secretions.

Cope and Zachary (11) report a case in which they credit success to a high enterostomy:--

F. B., a man, aged 28, was taken with acute abdominal symptoms on March 18, 1924. When first seen on March 21, he was clearly suffering from acute appendicitis. At the Bolingbroke Hospital on that date a very acutely inflamed and gangrenous appendix which lay against the right wall of the pelvis, under cover of a hood of inflamed omentum. The contaminated omentum was removed, together with the appendix, and drainage by rubber tube instituted. All went well for three days, but on March 24 the patient began to vomit copious quantities of fluid, which soon became faeculent. There was no pain and the pulse remained at 88, but collapse soon supervened; the cheeks became sunken, the pupils dilated and it was evident that no time should be lost in remedying the obstruction. On re-opening the wound the last coil of the ileum was found obstructed and adherent

to the right wall of the pelvis. It appeared difficult to clear it, so enterostomy, with short-circuit of the base of the obstructed loop of gut, was carried out. Next morning the patient was much better, and the enterostomy had acted well, but later in the day the flow almost ceased. Vomiting re-started, and the patient again became collapsed. The abdomen was now opened higher up and enterostomy performed in a coil of jejunum. At this operation it was noted that the lower part of the small intestine was enormously thickened with inflammatory edema, and an attempt was made to open the gut above the inflamed area. There was no evidence of peritonitis. In order to lessen the amount of fluid lost, the suggestion put forward by Wilkie was adopted, and the upper and lower enterostomies were connected by tubing which permitted the fluid escaping from the one opening to re-enter the bowel by the other. For three days the patient's condition improved, but on March 28 the upper tube ceased to drain well, and the patient vomited pints of bile and again looked almost moribund.

The upper wound was then re-opened, and it was found that the thickened edematous condition had spread farther up the small intestine, beyond the enterostomy. The second enterostomy was therefore closed, and a third one made still higher up at a point where the jejunum was closely in contact with a perfectly healthy looking transverse colon.

It was specially noted that there was no peritonitis, and the contrast between the distended, very thickened jejunum and a contracted, normal looking colon which was lying adjacent to it was very striking. To make drainage of the small bowel more complete a portion of the upper jejunum was also anastomosed to the transverse colon. The stomach was also washed out.

At each of these three operations two pints of saline were given intravenously to combat the shock. At no period did the patient complain of and pain. From that time improvement began, and after a prolonged convalescence, during which the enterostomies were closed, recovery took place. A recent x-ray photograph shows that there is no obstruction, though attacks of colicky pain arise occasionally.

It is interesting to note that the different operations gave the authors opportunity to observe an ascending inflammation of the small bowel, involving the whole thickness of the bowel wall, and gradually ascending almost to the duodeno-jejunal junction. There was no peritonitis, for the large bowel was clear. A draw back which may be attributable to this short-circuit is that the man now has a portentous appetite, and the bowels are opened rather soon after a meal.

11. Southam (57) believes that bile administered by rectum--two ounces in four ounces of water--diminishes symptoms. The full value of this is doubtful.

12. Nourishment should be administered principally in the

form of glucose per rectum.

The disturbances of the intestinal function as occurs following surgical interventions, especially after laparotomies, but also in the course of other intraperitoneal or extraperitoneal diseases are very much feared complications and have been the cause of the most varied therapeutical suggestions. Fortunately, however, the condition is much less frequently seen, resulting principally from more effective surgery and prophylactic measures and it is in this direction that stress should be laid. In the future there will undoubtedly be fewer and fewer cases. Many of the reflex cases however, as caused by pyelitis, trauma, etc., shall always demand immediate treatment and a thorough understanding of the condition by every physician is needed. Undoubtedly the future holds many enlightening discoveries in this respect; especially in reference to etiology and treatment.

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